UNITED STATES DISTRICT COURT
DISTRICT COURT OF NEVADA

LaKISHA NEAL-LOMAX, JOSHUA WILLIAM LOMAX, ALIAYA TIERRAEE LOMAX, JUANITA CARR, as parent and guardian of INIQUE ALAZYA LOMAX, and JOYCE CHARLESTON, individually, and as Special Administrator of the Estate of WILLIAM D. LOMAX, JR.,

Plaintiffs,

vs.

LAS VEGAS METROPOLITAN POLICE DEPARTMENT; OFFICER REGGIE RADER, in his individual and official capacity; SHERIFF BILL YOUNG, in his official capacity; TASER INTERNATIONAL, INC., an Arizona Corporation; TASER INTERNATIONAL, INC., a Delaware Foreign Corporation; DOES I through X; DOES XI through XX; and ROE CORPORATIONS XXI Through XXX, inclusive,

Defendants.

Case No.CV-S-05-01464-PMP-RJJ

Expert Report: Vincent J.M. Di Maio, M.D.
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Pursuant to Fed. R. Civ. P. 26(a)(2), I, Vincent J.M. Di Maio, hereby submit my report that contains a complete statement of all opinions to be expressed and the bases and reasons therefore; the data and other information I considered in forming the opinions; the exhibits or list of references I used as a summary of or support for the opinions; my qualifications, including a list of all publications authored within the preceding ten years; the compensation to be paid for the study and testimony; and a listing of any other cases in which I have testified as an expert at trial or by deposition within the preceding four years.

Vincent J.M. Di Maio

April 19, 2007
Date

INDS02 MCR 884019v1

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April 16, 2007

As requested, I have reviewed the four books of material sent me in regard to the aforementioned case. Attached is the Index of the volumes.

William Lomax was a 26 year-old black male who, on the afternoon of 2/20/04, became involved in an altercation with housing authority security guards at the Emerald Gardens Apartment Complex in Las Vegas, Nevada. The guards approached him because of bizarre behavior manifested by irrational outbursts, impaired cognition and combativeness. There had been a similar encounter with Mr. Lomax on 12/17/03 due to his use of PCP (phencyclidine). At that time, he had been sent to University Medical Center where he admitted to use of PCP and demonstrated a sympathomimetic toxidrome manifested by tachycardia, hypertension and diaphoresis. As in the previous incident, the guards requested assistance from the police and medical personnel.

Officer Reggie Rader of the Las Vegas police department, who was at the complex for another call, saw the encounter between the guards and Mr. Lomax and went to their assistance. Officer Rader used his Taser in the stun mode in order to assist the guards in their efforts to handcuff Mr. Lomax. The Taser was applied to the base of the neck and activated approximately two times for 3 and 4 seconds, respectively. Two handcuffs attached end to end were then used to handcuff Mr. Lomax's hands behind his back. The witnesses at the scene stated that while use of the Taser would cause Mr. Lomax to cease his violent conduct, this reaction to its use was very transitory with rapid return to his aggressive behavior.

At approximately, 1758 hrs, a Las Vegas Fire Department engine arrived at the scene. The crew of four consisted of the senior officer, a firefighter, an EMT and a paramedic. The crew observed Mr. Lomax to be lying face down on the ground with his hands cuffed behind him with two pairs of cuffs. He was yelling, kicking and combative. Three guards and the police officer were attempting to hold Mr. Lomax down. The guard holding the right arm was applying pressue on Mr. Lomax's back with his knee. When subsequently ordered to stop this, he complied. Mr. Lomax continued to struggle and yell.

Shortly after the arrival of the engine unit, the AMR ambulance arrived. They made similar observations as the engine unit as to the condition of Mr. Lomax. The AMR personnel then removed a gurney from the unit and wheeled it to Mr. Lomax. Paramedic Ritz of AMR took out a Velcro restraint and attached it to Mr. Lomax's right arm. He gave Paramedic Pearson of the fire department a second restraint and the latter attached it to the left arm. Mr. Lomax was then

lifted onto the AMR gurney, and placed face down. At this time, he was agitated, yelling and combative. It was then decided to remove the cuffs and use soft restraints, i.e. the Velcro ties, to restrain him. During the struggle on the ground and then to put him on the gurney and replace the handcuffs with soft restraints Mr. Lomax was Tasered 5 times over a 2 minute period. The last two times, he was Tasered 2 and 6 seconds, respectively. After the last time, he became relatively docile and the cuffs were removed and he was tied to the gurney with the soft restraints. He offered resistance to placement of the Velcro wrist restraints according to Paramedic Ritz. A strap was also put across the legs. He was then assessed by paramedic Robert Pearson who determined he had a respiratory rate of 16 and a pulse of greater than 120 beats/m.

Mr. Lomax was then wheeled to the ambulance and placed on it. He was again assessed by paramedic Robert Pearson who determined he had a respiratory rate of 16 and a pulse of greater than 120 beats/m. A nasal cannula was inserted and an intravenous line started. He offered resistance to placement of the intra-venous line according to Paramedic Ritz. He was breathing when the nasal tube was inserted. He was then rolled over and found to be pulseless and apneic. This was a number of minutes after the last use of the Taser. An EKG showed asystole. Resuscitation was begun and he was transported to Valley Emergency Room arriving at 1850 hrs. The heart rate was restored but by this time he had anoxic encephalopathy. He subsequently developed acute renal failure, rhabdomyolysis and cardiac necrosis and was pronounced dead at 1325 hrs on 2/21/04. A toxicology screen was positive for PCP.

An autopsy was conducted at the Clark County Coroners Office on 2/22/04. The deceased weighted 233 lbs and was 70 inches tall. There were some minor external abrasions. Microscopic examination of the heart showed increased interstitial fibrosis and focal acute bronchpneumonia. Toxicological analysis of blood collected on 2/20/04 at 2000 hrs revealed a PCP level of 28 ng/ml while analysis of blood and liver collected at 1020 hrs on 2/22/04 revealed a PCP level of 129 ng/ml in the blood and 245 ng/mg in the liver.

Based on the aforementioned facts, it is my opinion that William Lomax died as the result of a fatal cardiac arrhythmia due to Excited Delirium Syndrome brought on by his use of the illegal drug PCP (phencyclidine). The increased interstitial fibrosis of the heart, which indicates prior injury to the heart, played a role in the death by making the deceased more susceptible to a cardiac arrhythmia. The presentation of asystole at the time of his cardio-pulmonary arrest rather than ventricular fibrillation is typical of deaths due to the Excited Delirium Syndrome. The microscopic pneumonia noted, in all medical probability, developed during his hospitalization secondary to his anoxic encephalopathy.

The use of the Taser did not cause this death. There is in fact no objective or scientific evidence that use of a Taser does cause death. The fact that he was restrained in the prone position also did not cause the death. Respiration in the prone position is more effective than in the supine position and use of restraints does not decrease the oxygenation of blood.

PCP was developed in the 1950s as an intravenous anesthetic. It is a noncompetitive antagonist of the N-methyl-D-aspartate (NMDA)/glutamate receptors. Use in humans was discontinued in

1965 because patients often became agitated, delusional, and irrational while recovering from its anesthetic effects. It is snorted, smoked, or ingested. The physical effects of its use include a rise in blood pressure and pulse rate, flushing, profuse sweating, and numbness of the extremities and loss of muscular coordination. Psychological effects mimic the symptoms of schizophrenia: delusions, hallucinations, paranoia, disordered thinking, and violent behavior. Repeated use of PCP may produce persistent symptoms of schizophrenia: psychosis, hallucinations, delusions, thought disorders, cognitive dysfunction and flattened affect. Symptoms can persist up to a year after stopping PCP use.

Excited Delirium Syndrome (EDS) involves the sudden death of an individual, during or following an episode of excited delirium, in which an autopsy fails to reveal evidence of sufficient trauma or natural disease to explain the death. In virtually all such cases, the episode of excited delirium is terminated by a violent struggle with police or medical personnel, and the use of physical restraint. In occasional cases, there may be use of Pepper Spray or a Taser in an attempt to control the individual.

Shortly following cessation of the struggle, the individual is noted to be in cardio-pulmonary arrest. Attempts at resuscitation are usually unsuccessful. If resuscitation is "successful", the individual is found to have suffered irreversible hypoxic encephalopathy and death occurs in a matter of days. Typically, the only findings at autopsy are minor abrasions and contusions explainable by the struggle that preceded death, as was true in this case.

The cause of death in cases of EDS is usually multifactoral, due primarily to a hyper-adrenergic state combined in some cases with the use of illegal stimulants, medications, natural disease and/or genetic polymorphism. In this case, death was due to activation of the Sympathetic System by the delirium, the struggle and use of PCP.

Deaths due to Excited Delirium were initially described in individual with mental disease, primarily schizophrenia and bipolar disease. The introduction of medication for mental disease in the early 1950's eliminated most such deaths. These deaths still occur in mental patients if the patient is off their medication. Most deaths of mental patients from EDS encountered by police are due to their stopping use of their medication and/or use of illegal stimulants.

Whenever one gets excited, such as in excited delirium, or engages in strenuous activity such as a struggle, or takes a hallucinogenic drug such as PCP, there is activation of the Sympathetic Nervous System with release of norepinephrine (NE) from nerve cells into the synaptic spaces between the Sympathetic neurons and receptor organs such as heart muscle and the coronary arteries. This causes the heart to beat harder and faster and to raise the blood pressure. At the same time, there is constriction of the coronary arteries with reduction of blood flow, and thus oxygen, to the myocardium.

Peak levels of catecholamines (norepinephrine and epinephrine) are reached not during the physical activity but in the 2-5 minutes after cessation of the activity and may reach 10x base levels. This is Dimsdale et al.'s "period of peril", when the heart is most sensitive to

development of fatal arrhythmias.

During the physical activity, blood potassium also increases. Elevated levels of catecholamines in the blood neutralize the arrhythmogenic potential of the elevated blood potassium. During the "period of peril", the blood potassium levels drop dramatically, at times to hypokalemic levels. Hypokalemia, like hyperkalemia, is arrhythmogenic, but its' effects are not protected by elevated blood catecholamine levels. Hypokalemia predisposes to prolongation of the QT-interval, development of *torsade de pointes* and sudden cardiac death.

Thus, anyone engaging in a struggle or strenuous activity, after cessation of the struggle, enters a "period of peril" characterized by peak levels of catecholamines and dramatically falling levels of potassium. While the usual result of these physiological changes is uneventful with a complete return to normal, in some individuals, especially those in excited delirium (ED), death can result.

In regard to the plaintiff's expert witness reports, a number of erroneous assumptions and conclusions are present. Thus, the mechanism of death is said to be respiratory arrest, though the facts disprove this. Mr. Lomax was observed to be breathing up to the time he arrested. For respiratory arrest to have caused the hypoxic encephalopathy, the brain would have had to been deprived of oxygen for 5-8 minutes. Thus Mr. Lomax would have had to have been in respiratory arrest for that period of time - which he was not. The effects of short term respiratory arrest would have been reversed by ventilation. The oxygen deprivation of the brain was due to cardiac arrest with lack of perfusion of the brain in spite of attempted cardiac resuscitation.

The experts contend that the prone position of Mr. Lomax with his hands cuffed behind his back caused hypoxia. In fact clinical research indicates that gas exchange is improved by the prone position and binding the hands behind the back when coupled with hogtying (a situation much more incapacitating than occurred in this case), produces only minor changes in ventilatory functions and, more importantly, no change in oxygenation of the blood.

The microscopic pneumonia noted at autopsy, in all medical probability, played no role in Mr. Lomax's cardio-pulmonary arrest but rather developed during his hospitalization secondary to his anoxic encephalopathy. Comments about neurological damage from the electrical current generated by the Taser are sheer speculation with absolutely no foundation. The references to permanent neurological damage in the literature refer to cases of electrocution.

Based on the aforementioned facts, it is my opinion that, in all medical probability, William Lomax died as the result of the Excited Delirium Syndrome with the excited delirium due to use of the illegal drug PCP. The mechanism of death was a hyperadrenergic state produced by the excited delirium, and the struggle. The increased interstitial fibrosis of the heart, which indicates prior heart injury, played a role in the death by making the deceased more susceptible to a cardiac arrhythmia. There is no scientific evidence that the use of the Taser caused or contributed to the death.

I am a physician Board Certified in Anatomical, Clinical and Forensic Pathology. Attached is my

Curriculum Vitae which gives details of my education, qualifications, professional experience and publications. I have testified in state and federal courts throughout the United States as well as in courts in Canada and South Africa. Also attached is a list of cases that I have testified in and a fee schedule. I reserve the right to amend this report should additional information be presented for my review.

Sincerely,

VINCENT J.M. DI MAIO, M.D.

CURRICULUM VITAE

NAME:	Vincent J.M. Di Maio
ADDRESS:	5 Reading Lane San Antonio, Texas 78257 (210) 698-1400 vincent_dimaio@yahoo.com
BIRTHDATE:	March 22, 1941
BIRTHPLACE:	Brooklyn, New York
COLLEGE:	St. John's University, 1958-1961
MEDICAL SCHOOL	State University of New York Downstate Medical Center, 1961-1965

POSTGRADUATE TRAINING

Internship in Pathology:	Duke University Hospital, Durham, NC July 1, 1965 - June 30, 1966
Residency in Pathology:	State University - Kings County Medical Center, Brooklyn, NY June 1, 1966 - June 30, 1969
Fellow in Forensic Pathology:	Office of the Chief Medical Examiner of Maryland, Baltimore, Maryland, July 1, 1969 - June 30, 1970

BOARD CERTIFICATION

American Board of Pathology, ANATOMICAL PATHOLOGY, 1970

American Board of Pathology, CLINICAL PATHOLOGY, 1970

American Board of Pathology, FORENSIC PATHOLOGY, 1971

MILITARY SERVICE

Major, Medical Corps, United States Army Reserve, July 1, 1970 - June 30, 1972. Assigned to Armed Forces Institute of Pathology, Washington, D.C.

PRESENT POSITIONS

Consultant in Forensic Medicine And Pathology	March 1, 1981 - present
Editor-In-Chief	American Journal of Forensic Medicine and Pathology, January 1, 1992, to present
Consultant	Bexar County Medical Examiner's Office, 1/01/07 to present
Member	Strategic Planning Committee of National Association of Medical Examiners, 2003 to present
Member	Working group to develop standards/guidelines for medical examiners, Standards, Inspections and Accreditation committee of the National Association of Medical Examiners
Board of Directors	National Association of Medical Examiners, 2004 to present

PRIOR POSITIONS

Chief Medical Examiner	Bexar County, San Antonio, Texas March 1, 1981, to December 31, 2006 (retired)
Professor	Department of Pathology, University of Texas Health Science Center at San Antonio, February 1, 1987, to December 31, 2006
Consultant	Saville Inquiry into "Bloody Sunday", 2003-2004
Member	Strategic Planning Committee of National Association of Medical Examiners, 1999 to 2001
Consultant	Assassination Records Review Board, Washington D.C.
Consultant	United Nations Office of the Prosecutor for the International Criminal Tribunal for the Former Yugoslavia (September 1997 - February 1998)
Director	Bexar County Criminal Investigation Laboratory San Antonio, Texas March 1, 1981 - April 15, 1997
Medical Examiner	Office of the Dallas County Medical Examiner, Dallas, TX, July 1, 1972 - February 28, 1981

Associate Professor	Dept. of Pathology, University of Texas Health Science Center at Dallas, September 1, 1977 - February 28, 1981
Assistant Professor	Dept. of Pathology, University of Texas Health Science Center at Dallas, September 1, 1974 - August 31, 1977
Instructor	Dept. of Pathology, University of Texas Health Science Center at Dallas, July 1, 1972 - August 31, 1974
Chief, Wound Ballistics Section	Forensic Pathology Branch, Armed Forces Institute of Pathology, July 1, 1971 - June 30, 1972
Chief, Legal Medicine Section	Forensic Pathology Branch, Armed Forces Institute of Pathology, Sept. 1, 1970 - June 30, 1971

PROFESSIONAL OFFICES

Board of Editors	American Journal of Forensic Medicine and Pathology, February 1980 -
Board of Editors	Legal Medicine (Japanese Soc. of Legal Med), 1999-

PRIOR PROFESSIONAL OFFICES

Board of Editors	Journal of Forensic Sciences, February 1980 - 2000
Board of Directors	National Association of Medical Examiners, 1980 - 1986
Chairman	Council on Forensic Pathology, American Society of Clinical Pathologists, 1979 - 1982
Member	Council on Forensic Pathology, American Society of Clinical Pathologists, 1976 - 1982
Editor	Forensic Science Gazette, Sept. 1, 1974 December 31, 1980
Editorial Board	Pathologist, College of American Pathologists, 1980 - 1983
Consultant	Department of Pathology, Baylor University

Medical Center, Dallas, Texas,
July, 1980 - February, 1981

PROFESSIONAL MEMBERSHIPS

Fellow, American Academy of Forensic Sciences

National Association of Medical Examiners (1971 - present)

Membro academico correspondiente Academia de Medicinia legal y ciencias forenses de la Republica Argentina

AWARDS

"The CCE Commissioners' Medal" by The American Society of Clinical Pathologists

The "Jean R. Oliver, M.D. Master Teacher Award", presented by the Alumni Association of the State University of New York-Downstate Medical Center. Brooklyn, New York, May 12, 1990

"The George E. Gantner Jr., Memorial Award" presented by the National Association of Medical Examiners, Baltimore, MD, September 16, 1997

"Outstanding Service Award" presented by the National Association of Medical Examiners, Minneapolis, Minn. October 19, 1999

"Milton Helpern Award" Presented by the Pathology/Biology Section American Academy of Forensic Sciences Chicago Illinois, February 19, 2003

Milton Helpern Laureate Award Presented by the National Association of Medical Examiners, October 17, 2006

BOOKS

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3. Di Maio, V.J.M. **Gunshot Wounds** - Practical Aspects of Firearms, Ballistics and Forensic Techniques. 2nd ed. CRC Press Inc., Boca Raton, FL, 1999.

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4. Di Maio, V.J.M. and Dana, S.E. **Handbook of Forensic Pathology.** 2nd ed. CRC Press Inc., Boca Raton, FL, 2006

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5. Di Maio, V.J.M. (Editor) Symposium on Forensic Pathology - Clinics in Laboratory Medicine. Vol. 3, No. 2, June 1983, W.B. Saunders Co., Phil, PA.

ARTICLES

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2. Minkowitz, S., Zeichner, M., DiMaio, V.J.M. "Cystosarcoma Phyllodes: A Unique Case with Multiple Unilateral Lesions and Ipsilateral Axillary Metastasis", J. Path. Bact., 96:514-517, 1968.

3. DiMaio, V.J.M., Spitz, W.U. "Injury by Birdshot", J. Forensic Sci., 15:396-402, 1970.

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5. DiMaio, V.J.M., DiMaio, D.J. "A Postpartum Dissecting Coronary Aneurysm", NY State J. Med., 71:767-769, 1971.

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7. DiMaio, V.J.M., DiMaio, D.J. "Bullet Emboli: Six Cases and a Review of the Literature", J. Forensic Sci., 17:394-398, 1972.

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9. DiMaio, V.J.M. "Wound Ballistics", J. Assoc. Firearms/Toolmark Examiners, 4:27-29, 1972.

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23. DiMaio, V.J.M., Minette, L.J., Johnson, S. "Three Deaths due to Revolver Shot Shell Cartridges", Forensic Sci., 4:247-252, 1974.

24. DiMaio, V.J.M., Garriott, J.C., Putman, R. "Digoxin Concentration in Postmortem Specimens after Overdose and Therapeutic Use", J. Forensic Sci., 20:340-347, 1975.

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Skin", J. Forensic Sci., 21:367-372, 1976.

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2. DiMaio, V.J.M., "Summary of Remarks", in Wound Ballistic Workshop Presentations, FBI Academy, January 19 - 22, 1993

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FEE SCHEDULE

Review of materials; conferences – per hour	$ 400.00
Deposition taken out of San Antonio	$ 3,200.00 Plus expenses & travel time
Deposition in San Antonio per hour	$ 500.00
Court Testimony (one day or part thereof)	$ 3,200.00 Plus expenses & travel time
Travel time/waiting time – per day	$ 1,600.00*
per hour	$ 200.00 Plus expenses
Retainer	$ 2,400.00

* Air travel outside Texas is to be First Class

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NOTE: Most of the cases that I testify in are Bexar County cases. They involve individuals that I have autopsied in my job as Chief Medical Examiner. My Office does not keep a list of these cases nor does the District Attorney.

Cases where I testified or gave a deposition (1994-2005):

Hawaii v. Graham DA Office	Asphyxia, homicide
Ching v. City and County of Honolulu	Allegedly beaten by Officer
LaCorte v. Honolulu et al	Individual shot by Officer
Ramirez vs. Vanguard Care Center David Marks, Houston	Decubitus ulcer/ Nursing Home case
Smith v. Olin Eppenberger, St. Louis, Missouri	Accidental gunshot wound
Az v. Copeland	Stabbing homicide
Az v. Carpenter Phoenix DA	CCI homicide
Bryant vs. Fost Joseph Turner, Austin TX	Drowning
Illinois v. Becker State's Attorneys Office, Chicago	Police shooting - criminal case
Florida vs. O'Connor State Attorney, Ft. Lauderdale	Police shooting - criminal case
Florida vs. W. K. Smith Roy Black, Miami Florida	Alleged rape

Gossett vs. City of Mansfield Bill Lane, Ft. Worth, TX	Police shooting
LeGue vs. Prysi Florida	Breast implant death
Osborne vs Arave	
Texas vs Routier	Stabbing
Texas vs Belton DA, Austin	Homicide, blunt force abdomen
Texas v. Kellum Kevin Brooks, Dallas	Alleged child abuse
Tx v. McBride	Homicide
Tx vs Randall Public Defender, Dallas	Alleged child abuse
Tx vs Mowbray (Cameron County)	Shooting - homicide
Wyom v Frias	Suicide vs homicide
Colorado v. Bayliss	Homicide, shooting
Colorado v. Baca	Homicide , shooting
Jones vs Father Walter Mem. Child Care Center Cunningham, Bounds, Yance, Crowder & Brown Mobile, Alabama	Traumatic asphyxia
Animashaun v. O'Donnell et al City of Chicago,	Cocaine related death
Virginia v. Roy Smith	Death sentence appeal
Ortega vs. GMC Hartline, Dracus, Dreyer & Kern Dallas, Texas	Traumatic asphyxia
Rentetia v. Garcia City of Chicago	
Low v. American Transitional Hosp. Marshall & Gonzalez, Houston, Texas	Alleged drug overdose

Nevada v. Middleton	Serial murderer
Marty vs. GM Hartline, Dracus, Dreyer & Kern Dallas, Texas	Motor vehicle accident
Penn. v. Dillon	Homicide
Commonwealth. V. Clark Mass.	Homicide, shooting
Pennsylvania v. Scher Office of the Attorney General	Shooting, homicide
NJ v. Speth Arseneault & Krovatin - Chatham, NJ	Tampering with evidence
People (Calif) v. Hasson DA, Riverside, CA	Homicide disguised as suicide
Patterson et al v. Horizon Health Care McLean & Sanders - Ft. Worth, Texas	Nursing home case
Sanchez v. Remington Rodriguez, Colvin & Chaney, Brownsville, Texas	Accidental gunshot wound
Zamora et al. V. Public Utilities Board of the City of Brownsville Anthony Constant, Corpus Christi	Electrocution
Estate of Courtney LaFlesch - Montana Sol & Wolfe, Attorneys, Missoula, Montana	Domestic shooting
Harrison vs Lee Lewis et al Carl Crow, Houston, Texas	Relation of alcohol to an accident
Morris et al. V. Archer-Daniel-Midland Carl Crow, Houston Texas	Relation of alcohol to an accident
Sanchez v. Daisy Ernesto Gamez, Brownsville, Texas	BB wound of eye
Smith v. YMCA Reynolds & Pennington, Ft. Worth, Texas	Drowning
Bird v. GMC Snell & Wilmer, Irvine, CA	Motor vehicle accident

Lewis vs. Four Seasons Fulbright & Jaw.... San Antonio	Insulin overdose in Nursing Home
Thibodeaux et al. V. Grocers Supply O'Quin & Laminack Houston, TX	Motor vehicle accident
Beckwith et al. V. Coyotes Entertainment Law Offices of Michael Park Huntsville, TX	Motor vehicle accident
Iowa vs Watson	Shotgun homicide
Nebraska v. Kula	Rifle homicide
Ealim v. Mazda	Motor vehicle accident (seatbelt)
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Texas vs. Tyson	Child homicide vs Tylenol death
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California v. Jacobs San Diego DA's Office	Strangulation (trial)
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Dick DeGuerin
Houston, Texas

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Office of the City Attorney	Excited Delirium Syndrome
Thompson v. Alza Corp et. al. Wright, Brown & Close Houston, Texas	Fenatnyl patch case

UNITED STATES DISTRICT COURT
DISTRICT OF NEVADA

LaKISHA NEAL-LOMAX, JOSHUA WILLIAM LOMAX, ALIAYA TIERRAEE LOMAX, and JOYCE CHARLESTON, individually, and as Special Administrator of the Estate of WILLIAM D. LOMAX, JR., Plaintiffs, vs. LAS VEGAS METROPOLITAN POLICE DEPARTMENT; OFFICER REGGIE RADER, in his individual and official capacity; SHERIFF BILL YOUNG, in his official capacity; TASER INTERNATIONAL, INC., an Arizona Corporation; TASER INTERNATIONAL, INC., a Delaware Foreign Corporation; DOES I through X; DOES XI through XX; and ROE CORPORATIONS XXI Through XXX, inclusive, Defendants.	)	Case No. A513602 Dept.: II

INDEX OF EXPERT BINDERS

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1) Transcript of Hearing in Inquest of William Lomax (obtained from Clark County Coroner)

2) Three CDs of 911 Calls, Autopsy Photos and TASER Information (obtained from the Clark County Coroner's office)

3) Other documents obtained from the Clark County Coroner's office (less officer statements)

4) Valley Hospital Medical Records

Binder 2

5) Voluntary Statement of Lorin Spendlove

6) Voluntary Statement of Brandon Israel

7) Voluntary Statement of Robert Pearson

8) Voluntary Statement of R. Rader

9) Voluntary Statement of Kelly Hintsala

10) Voluntary Statement of Joseph R. Herrera

11) Voluntary Statement of James Hines

12) Voluntary Statement of David Wireman

13) Voluntary Statement of Bryan Cornell

14) Voluntary Statement of Jason Ritz

15) Voluntary Statement of Keith Murray

16) LVMPD Use of the TASER Policies

17) LVMPD Use of Force Policies

18) LVMPD Standardized Lesson Plan for X/M-26 TASER User Course (11/25/03)

19) TASER Power Point - Certification Course - TASER X26 and M26 (Version X)

20) LVMPD Training Bureau - Executive Summary - Study of TASER Use with OC Spray

21) TASER X26 and M26 Certification Test

22) LVMPD Communication Center Report

23) Use of Force Written Reports by Officers

24) Incident Reports

25) Crime Scene Report

26) Research Compendium

27) Incident Recall (Dispatch Records)

28) TASER Device Download Data

29) American Medical Response Records (Responding Paramedic Records)

30) Clark County Fire Department Records (Responding Paramedic Records)

31) Lomax's Criminal Records

32) University Medical Center Records

33) Timeline of Key Events (To be provided by TASER Int'l)

Depositions (only being sent to medical experts)

34) Deposition of Jean Charleston (10/18/06)

35) Deposition of Joyce Charleston (10/18/06)

Binder 3

36) Deposition of Brandon Israel (8/2/06)

37) Deposition of Keith Murray (10/2/06)

38) Deposition of Robert Pearson (8/2/06)

39) Deposition of Michael Perkins (8/3/06)

40) Deposition of Jason Ritz (9/13/06)

41) Deposition of Lorin Spendlove (8/2/06)

42) Deposition of David Wireman (8/3/06)

43) Deposition of Reggie Rader (to be provided shortly)

44) TASER Training CD 11.0

45) X26 Operating Manual

46) LVMPD Standardized Lesson Plan

47) Updated Electrical Characteristics of TASER Devices

48) Clip from Deposition of Officer Reggie Rader

49) Draft of the deposition transcript of Dr. Ronald Knoblock in *Tucker v. Las Vegas Metropolitan Police Department, et al.*, United States District Court, District of Nevada

50) Excerpts from the Deposition of Dr. Ronald Knoblock

51) Excerpts from Paramedic Testimony

UNITED STATES DISTRICT COURT
DISTRICT COURT OF NEVADA

LaKISHA NEAL-LOMAX, JOSHUA WILLIAM LOMAX, ALIAYA TIERRAEE LOMAX, JUANITA CARR, as parent and guardian of INIQUE ALAZYA LOMAX, and JOYCE CHARLESTON, individually, and as Special Administrator of the Estate of WILLIAM D. LOMAX, JR.,

Plaintiffs,

vs.

LAS VEGAS METROPOLITAN POLICE DEPARTMENT; OFFICER REGGIE RADER, in his individual and official capacity; SHERIFF BILL YOUNG, in his official capacity; TASER INTERNATIONAL, INC.; an Arizona Corporation; TASER INTERNATIONAL, INC., a Delaware Foreign Corporation; DOES I through X; DOES XI through XX; and ROE CORPORATIONS XXI Through XXX, inclusive,

Defendants.

Case No.CV-S-05-01464-PMP-RJJ

Expert Report: Dr. Dorin Panescu
5275 Country Forge Lane
San Jose, CA 95136
Phone: 408-522-6372

Pursuant to Fed. R. Civ. P. 26(a)(2), I, Dorin Panescu, hereby submit my report that contains a complete statement of all opinions to be expressed and the bases and reasons therefore; the data and other information I considered in forming the opinions; the exhibits or list of references I used as a summary of or support for the opinions; my qualifications, including a list of all publications authored within the preceding ten years; the compensation to be paid for the study and testimony; and a listing of any other cases in which I have testified as an expert at trial or by deposition within the preceding four years.

Dorin Panescu

April 19, 2007
Date

INDS02 MCR 884020v1

1. Introduction and Summary of Findings

1.1 Intent of the report and Dr. Dorin Panescu's credentials

The intent of my report is to provide opinions regarding to the probable distribution of electrical currents produced by TASER electronic control devices (ECDs or devices) in the human body and whether TASER electrical currents could have had a causal role in the death of Mr. William Lomax.

Throughout my 20-year career, I have conducted research, developed, invented and published about medical electrical devices, particularly cardiac devices. Appendix A includes a copy of my Curriculum Vitae, summarizing my education, experience, patents and publications. Appendix B lists the cases I have been involved with as an expert witness and my hourly compensation.

1.2 Summary of Findings

After reviewing the evidence presented in Section 2, I arrived at the following findings:

1.2.1 The X26 TASER device generates significantly less charge and energy than other medical devices, such as external defibrillators and ablation RF generators, that are approved and deemed safe for medical use. Theoretical risks associated with the use of TASER devices are far lower than those accepted by regulatory agencies, such as the United States Food and Drug Administration (FDA), for approved medical devices.

1.2.2 The electrical charge carried by the first, also the highest, current peak of the X26 TASER device is, by a wide margin, significantly below charge-based thresholds known to be capable of inducing ventricular fibrillation (VF).

1.2.3 When applied in drive-stun mode to superficial neck area locations, only non-dangerous fractions, if any, of the voltage, current and charge generated by the X26 TASER reach the heart.

1.2.4 When applied in drive-stun mode to superficial neck area locations, current and charge generated by the X26 TASER can capture local motor nerves and cause temporary loss of neuromuscular control, with the initial reaction being a gravitational dysreflexia. Thereby, it can be effective in achieving temporary suspect incapacitation, as per intended use.

1.2.5 When applied in drive-stun mode to superficial neck area locations, current and charge generated by the X26 TASER have a very small and unlikely chance of reaching deeper layers, such as the phrenic nerve. With a high degree of medical probability, the residual electric field strength in layers, such as the phrenic nerve, is not high enough to produce any permanent damage. For example, electric field strength thresholds required for irreversible electroporation are not reached.

1.2.6 The concept of delayed ventricular fibrillation following electrical injury is not backed by any scientific evidence. To the contrary, long-term follow-up studies report the lack of delayed VF or dangerous arrhythmias.

1.2.7 Reports filed in this case show that Mr. Lomax confronted and struggled with housing security officers, fire personnel, paramedics and a police officer, was physically violent, did not comply with officers' orders, had several X26 TASER applications in the neck area, was overdosed with phencyclidine, had cannabinoids in his system, had mild tachycardia, high blood pressure, respiratory impairment, and, ultimately, passed away on February 21, 2004.

1.2.8 Phencyclidine (PCP) is a dissociative anesthetic that was originally synthesized for intravenous use in 1957. If abused, phencyclidine may cause serious adverse reactions, such as extreme agitation, high blood pressure (including complications, such as intracerebral complications), mild tachycardias, cardiac muscle fibrosis, psychosis, nystagmus, tachypnea and irregular respiratory, bronchorrhea, rhabdomyolysis, muscle rigidity, dystonia, opisthotonos, torticollis, akinesis, and hyperkinesis.

1.2.9 Mr. Lomax had a documented history of phencyclidine and cocaine abuse. On the day of his death, he reportedly exhibited most of the overdosing symptoms and conditions listed above. The autopsy report indicates phencyclidine concentrations as high as 245 ng/ml. A phencyclidine dose of 1000 ng/ml is considered lethal.

1.2.10 Dr. Bush's report, in spite of stating that 8% of PCP intoxications result in fatalities, seems to rush to dismiss PCP as having a proximate role in Mr. Lomax's death. No analysis is provided to elucidate scientifically whether reported symptoms, such as mild tachycardia, high blood pressure, cardiac fibrosis, extreme agitation, loss of orientation, respiratory difficulties, were related to Mr. Lomax's abuse of PCP.

1.2.11 Dr. Strote's report states that the TASER currents may have disrupted Mr. Lomax's respiratory system. Dr. Strote goes on to speculate that stun mode application in the neck area caused contraction of accessory muscles. However, he does not present evidence for his statement. The finite element analyses presented later in this report show that the temporary incapacitation achieved by TASER devices does not involve direct muscular capture. Rather, TASER currents capture motor nerves that in turn cause loss of neuromuscular control. As such, Dr. Strote's assumption that TASER discharge captured respiratory accessory muscles is speculative at best.

1.2.12 Dr. Woodard's report states that TASER applications worsened Mr. Lomax's already present metabolic derangement. However, Dr. Woodard does not provide any scientific quantification for the amount of additional derangement. Consequently, his conclusion that the TASER application contributed to Mr. Lomax's expiration is vague, made without scientific backing and is speculative.

1.2.13 Dr. Rhode's report shows a misunderstanding of the "skin effect" as applied to TASER currents. Additionally, Dr. Rhode makes speculative statements regarding nerve damage produced by TASER currents. Particularly, Dr. Rhode speculates that TASER currents could produce phrenic nerve damage. He provides no scientific base for his statements. Dr. Rhode uses his speculative nerve damage statements to make a point that TASER current caused Mr. Lomax to have a respiratory arrest which resulted in his death. The electric field analyses later in this report show that, with a high degree of medical probability, phrenic nerve damage is very unlikely with use of TASER devices. Corroborating the electric

field analyses with the autopsy results, which show no evidence of phrenic nerve damage, dismisses Dr. Rhode statements and reduces his conclusion to baseless and unfunded speculation.

2. Evidence of Materials Considered

In preparing the report, I have considered the following references and evidence:

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2 Voluntary statement Brandon Israel.
3 Voluntary statement by and 3/15/2007 videotaped interview with Officer R. Rader.
4 Incident Reports
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7 University Medical Center Records
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3. Findings and Opinions

3.1 Background

According to [1-5], on February 20, 2004, Mr. Lomax entered a conflict with housing officers. He was asked if he needed medical care and replied affirmative. Officers indicated he had symptoms of phencyclidine intoxication. While waiting for medical services to arrive, Mr. Lomax became confrontational and physically violent. Officer Rader gave Mr. Lomax multiple verbal orders to comply and to stop his physical aggression. As a result of not complying with officer's orders, Mr. Lomax's had several X26 TASER applications in drive-stun mode in the neck area. Mr. Lomax was finally restrained but continued to struggle. When emergency medical personnel arrived, an attempt was made to place Mr. Lomax on a gurney for transportation reasons. As he continued being confrontational, Officer Rader applied drive-stun X26 TASER discharges again. Mr. Lomax was then transported to the ambulance, while still being agitated. While in the ambulance, at some time after the last TASER discharge, Mr. Lomax became unresponsive. His rhythm was documented as asystole. In spite of being asystolic, medical personnel incorrectly applied defibrillation shocks to Mr. Lomax chest. External pacing was later applied, with a significant delay though. This delay in applying the appropriate emergency therapy may or may not have had critical effects on the lack of final recovery experienced by Mr. Lomax. Resuscitation efforts continued and, at the hospital, cardiac function returned. On February 20, 2004, at 20:51, his rhythm was mildly tachycardic, documented at 123 beats per minute (bpm), his systolic blood pressure was mildly elevated at 141 mmHg, with a respiratory rate of 20 [6]. These numbers are consistent with Mr. Lomax's PCP overdosage [7-8]. His medical history records indicate prior abuse of PCP with similar symptoms that required medical intervention [7]. In spite of cardiac function return, Mr. Lomax did not have signs of neurological recovery, as evidence by a brain flow scan [6]. He was declared dead on February 21, 2004 [6].

The autopsy report, [8], concludes that Mr. Lomax "died as a result of cardiac arrest during restraining procedures. Phencyclidine intoxication and bronchopneumonia were significant contributing conditions." Additionally, the report finds that Mr. Lomax had pulmonary congestion and edema, foci of increased interstitial cardiac fibrosis and PCP concentrations as high as 245 ng/ml, compared to lethal dosage of 1000 ng/ml [8]. The report does not list any evidence of respiratory arrest, no musculoskeletal abnormalities (e.g. as in those that could have been theoretically produced by too strong electric fields), no damage to anterior neck muscles, no focal areas of pathological changes to the central nervous system (CNS) structures, unremarkable brain vessels, no CNS hemorrhage [8].

3.2 Probable effects on the human body of electrical currents generated by TASER devices

3.2.1 Electrical output characteristic of X26 TASER device

As described in [9-13], the output of a TASER X26 ECD is characterized by peak arcing voltages of about 50 kV, an internal energy of about 0.36 J/pulse, at a rate of 19 pulses/s and an internal electrical power level of about 7 W. After the initial arc, the peak voltage becomes approximately 1.2 kV (or 400 V average over the duration of the main phase), with a delivered energy per pulse of about 0.07 J/pulse. Only about 1.3 W of electrical power are delivered externally. The total active waveform duration of 100 μs and delivers a charge of approximately 100 μC. By comparison, some external defibrillator devices, medically approved to resuscitate patients, put out peak voltages in the 2-5 kV range, peak currents well in excess of 20 A, with durations of typically 5 ms. The total output energy usually exceeds 200 J [14-16]. Similarly, some of the FDA-approved cardiac or liver ablation radiofrequency (RF) generators have maximum output power ratings of 100 W, or more, and maximum output current ratings 1-2 A_{rms} [17, 18]. These generators can deliver power and current to the heart, or liver, for durations that exceed 60 s. These outputs are equivalent, or at least an order of magnitude larger than the output of X26 TASER devices. Yet, such devices are approved for medical use and are widely believed to be safe in terms of not producing damage to cardiac structures.

3.2.2 Current distribution inside the human thorax and probable effects on the heart

Previous research I conducted on ventricular (de)fibrillation, defibrillation and pacing devices [15, 19-20], showed that even under optimal electrode placement configurations, only a low fraction of the current that entered the human thorax reached the heart. For example, we found that more than 66% of the input voltage dropped across portions of the thorax within 4 cm under cardiac electrodes that were optimally placed [15, 19]. For same optimal electrode cardiac placement, less than 10% of the input voltage dropped across the left ventricle [20]. The high resistances of the skin, the fat layer and the thoracic cage reduced the voltage gradient across the heart. Consequently, the current density at the heart level was significantly reduced with respect to values measured at electrode levels.

Koning presented that in order to successfully defibrillate a heart (defibrillation, the reverse of fibrillation, is a process whereby electrical currents resynchronize the cardiac cells) charge levels of at least 42 μC/g were required. The ratio is with respect to the mass of the heart [16]. For a heart of 320 g, such as Mr. Lomax's, 42*320 μC = 13,440 μC would be required for a successful defibrillation. By comparison, the charge delivered by an X26 TASER is less than 100 μC [9-13]. While defibrillation currents are usually larger than currents required for triggering fibrillation, still the ratio between required defibrillation charge and X26 charge, 13440/100, is greater than 134 times.

McDaniel *et al.* found that the blood pressure of animals stimulated with TASER devices was within normal range, an indication that no critical cardiac tachycardias took place [12, 13]. Furthermore, they found that more than 2000 μC of charge were required to fibrillate animals with a mass of 117 kg (about 250 lbs, compared to Mr. Lomax's weight of 233 lbs). This charge level represented a significant safety of margin with respect to the charge of the first pulse of the X26 TASER.

Deale and Lerman studied the ratio of transcardiac to transthoracic threshold currents in dogs [21]. They reported that the thoracic cage shunted 82% of the input

current and that the lungs shunted 14%. Only the remaining 4% of the input current passed through the heart.

The references cited above indicate that only a small fraction of the input current generated by external devices reaches the heart. Other layers of tissue, such as thoracic cage, fat, intercostal muscles, divert most of the current away from the heart. It is commonly accepted that the amount of charge deposited in myocytes is the main contributor to the onset of ventricular fibrillation. Work cited above shows that the charge generated by the X26 TASER is below fibrillation thresholds by a wide margin of safety. It is also important to emphasize that there are no significant changes noted in blood pressure levels during TASER applications [12, 13].

3.2.3 Review of X26 TASER electrical output with respect to requirements of standard IEC 479-1 and –2

The IEC 479 standard deals with effects of current on human beings and livestock [22, 23]. As stated in IEC 479-1, section 3, page 39, and section 4, page 49, describe the effects of sinusoidal alternating currents with frequencies between 15 Hz and 100 Hz and of direct currents passing through the human body, respectively [22]. The effects of non-sinusoidal currents of higher frequencies are covered by IEC 479-2. Section 4.4 describes the thresholds of ventricular fibrillation for impulses of short duration [23]. It states that "for 50% probability of fibrillation, Fq is of the order of 0.005 As." Fq is defined as the charge of the impulse. By the definition of current, charge and time units of measurement, the quantity 0.005 As is equal to 5000 μC. As presented in 3.2.1, the X26 TASER current (and by far the largest) carries a charge less than 100 μC. This is at least 50 times less than the threshold indicated by IEC 479-2 for a 50% probability of ventricular fibrillation induction.

3.3 Risk assessment of theoretical effects of TASER ECD currents

TASER International Inc. reported that ECDs were used in more than 232,000 human volunteer and 383,000 human suspects during actual law enforcement field deployments [24]. In any of these situations, no scientific or medical evidence was provided that TASER ECDs caused cardiac rhythm disturbances or neuromuscular or skeletal damage. As such, the overall critical risk of using TASER ECDs is estimated at less than 1/(232000+383000) = 0.0000016

3.3.1 EN 60601-1 rational for acceptable levels of VF risk

The EN 60601-1 international standard stipulates accepted regulatory requirements for the safety of electrical medical devices [25]. Particularly, this standard sets the allowed threshold for the patient leakage current for medical devices that have direct contact to patients' heart. Citing from the standard, we learn that [25]:

"The allowable value of PATIENT LEAKAGE CURRENT for TYPE CF APPLIED PARTS in NORMAL CONDITION is 10 μA which has a probability of 0.002 for

causing ventricular fibrillation or pump failure when applied through small areas to an intracardiac site.

Even with zero current, it has been observed that mechanical irritation can produce ventricular fibrillation. A limit of 10 µA is readily achievable and does not significantly increase the risk of ventricular fibrillation during intracardiac procedures."

This implies that under normal device operation, the allowed maximum patient leakage current is 10 μA_{rms}. Although a 10-μA_{rms} patient leakage current has a 0.002-probability of causing VF or pump failure in humans, the standards accepts this value as being safe. Regulatory bodies, such as the US FDA or the Germany-based TUV, certify electrical medical devices as being safe for use in intracardiac clinical procedures if they comply with the patient leakage current limit above. Intracardiac procedures carry the highest risk for patients. Therefore, by accepting requirements of EN60601-1, these conservative regulatory bodies, including the US FDA, accept that a probability of causing VF of 0.002 represents an extremely low risk. This FDA-accepted probability level of 0.002 is about 1250 times higher than the probability estimates for TASER device-induced risk estimated above.

3.3.2 Probabilities of risk encountered in common daily-life activities

A literature search on risk of daily-life activities showed that the average rate of car accident death in Italy in 1996 was 0.000219, while the rate of drowning in France in 1996 was 0.000016 [26]. Figure 1 tabulates certain risks per information collected by the World Health Organization [26].

	MALES ONLY, ALL AGES		
	France 1996	Italy 1996	UK 1997
Total deaths (000)	28,423	27,804	28,990
	Death per 100,000 per year		
Cardiovascular disease	280.2	401.2	430.0
Cancer	306.8	311.0	275.0
Cerebrovascular disease	63.5	102.7	85.9
Pneumonia	27.9	12.2	84.0
Diabetes	10.1	24.0	10.5
Nutritional deficiency	2.6	0.1	0.1
Accidents (all)	57.2	45.6	24.8
Road Traffic Accident	19.4	21.9	9.1
Drowning	1.6	1.3	0.7
Fire	1.1	0.7	1.1

(Based on mortality data in 'World Health Statistics, 1997-1999 edition. World Health Organization. http://www.who.int/)

Fig. 1. Risk probabilities in France, Italy and UK [26].

Similarly, another study showed that in the US about 5,700 pedestrians die every year while crossing the street [27]. Assuming a total of about 129 million pedestrians in the US, this number equates to a yearly compounded probability of dying while crossing the street of about 0.0000442. Other statistics help put things into perspective. For example, one study found that there is a probability of 0.116 of in-hospital death in patients with myocardial infarction that did not have early VF at the time of admission [28]. Another study presented that there is a probability of 0.018 of preoperative death in patients

admitted for implantation of cardiac stimulators [29]. Preoperative death occurs before any implantation procedure steps are taken.

Compared to the probability values listed above, the estimated theoretical upper limit of TASER critical risk, less than 0.0000016, is lower than the probability of death while crossing a street, or than that of dying while swimming and significantly lower than probabilities of death during car accidents or during certain medical procedures.

3.3.3. Risks associated with non-lethal weapons or use-of-force options available to law enforcement officers

Dr. Ho studied 162 in-custody death events [30]. He learned that in 68.5% of these cases, the suspects went hands-on with the law enforcement officers. In 100% of these cases, the suspects were handcuffed [30]. By comparison, TASER devices were involved in a lower 30.1% of these deaths. Obviously, it would be a stretch of imagination to think that because all suspects died while handcuffed, handcuffs could, therefore, cause cardiac arrest. There is no cause-effect relationship between the use of handcuffs and cardiac arrest, if any, in these suspects. Similarly, Dr. Ho found that in 0% of these deaths occurred within a short time after TASER deployment [30]. Corroborating Dr. Ho's finding with the risk assessment above, it results that it is highly likely, with reasonable degree of medical probability, that TASER ECDs are not contributory to cardiac arrest or to alleged in-custody deaths.

3.4 Probable effects of TASER currents on Mr. Lomax's heart

It is known that after the onset of ventricular fibrillation (VF) the blood pressure drops precipitously within a few seconds. As a result, the subject would lose consciousness within several seconds, certainly less than a minute, and also lose physical strength, control of gait and balance. Given the fact that Mr. Lomax reportedly had the physical strength to resist arrest even after the TASER device was used, it would be highly improbable that his heart experienced VF caused by currents delivered by the TASER device. The TASER device was applied in drive-stun mode to the neck area. Based on information presented in section 3.2.2 above, only an insignificant amount of current, if any, reached Mr. Lomax's heart. To further validate this statement, I developed a finite element model (FEM) of a male body, approximated on Mr. Lomax's relative physical attributes (177 cm actual height vs. 176 cm FEM length). Finite element modeling is a known mathematical technique that provides numerical approximations to solutions of differential equations, such as those governing electrical current distributions through the thorax [15]. The following tissue regions were modeled:

- Muscle (neck, shoulder, limbs)
- Bone (spine, ribcage)
- Heart
- Lungs
- Skin/Fat
- Abdomen

The model consisted of 8460 hexahedral elements. Tissue resistivities were assigned using values published in previous work [15, 19-20]. Figure 2 shows the FE mesh with its corresponding regions. Voltage type boundary conditions of 1000 V (approximate peak voltage of the X26 TASER) were assigned at nodes corresponding to approximate neck locations where the TASER electrodes touched Mr. Lomax. (Fig. 3) Per Officer Rader, the electrodes were placed in the collar bone area [3]. Although an approximation, I consider this voltage node placement representative of Mr. Lomax's actual situation.

Figure 2. Mesh of the finite element model.

Figure 3 illustrates the voltage distribution on the surface of the model. Note that voltage decreases rapidly with distance from the electrode location.

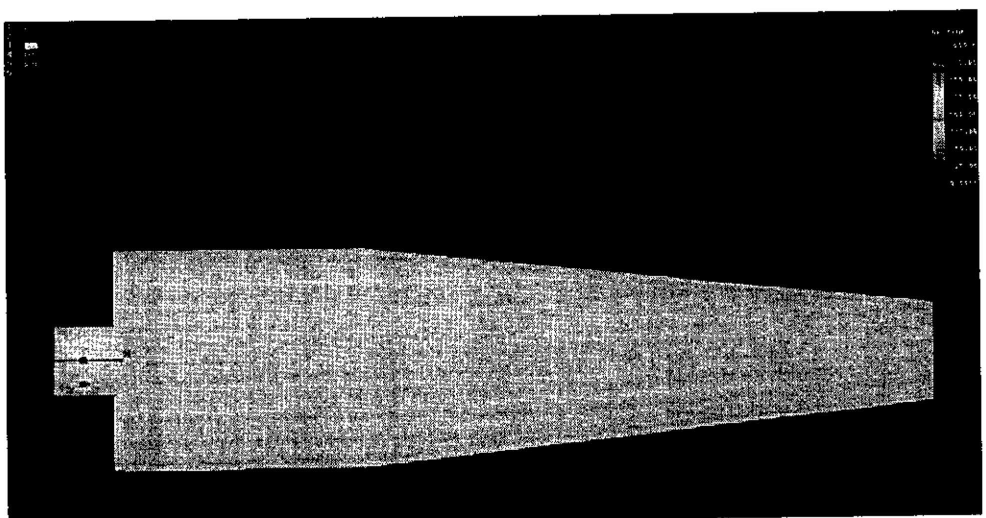

Figure 3. Voltage distribution on the surface of the FEM. This view also shows the location of the voltage boundary condition nodes.

Figure 4 illustrates through a model cross-section. The white rectangle indicates the heart volume. Figure 4 indicates that the current density decreases rapidly with distance from electrode. The maximum current density levels at the elements representing the heart is numerically estimated at 0.044 mA/cm^2. The FEM assumes an electrical resistivity of the heart region of 450 Ω·cm, situated at the higher end of values reported in the literature [15]. Based on these numbers, the corresponding maximum electrical field strength is approximately 0.02 V/cm (the electrical field strength is computed by multiplying the corresponding current density and resistivity values). The myocyte excitation threshold is reported to be between 2-5 V/cm [31-33]. Consequently, even considering the lower end of the interval, 2 V/cm, the current density values in the heart region, as predicted by the FEM for Mr. Lomax's circumstances, are significantly lower than the threshold required to initiate any kind of cardiac rhythm disturbances (at least about 100 times lower). It is important to note that current density levels required to induce sustained VF are much higher. Using rheobase and chronaxie numbers from a cited ventricular fibrillation model [34] provides a current density threshold for VF induction, prorated for the ~ 100-us duration of the X26 TASER pulse, of about 91 mA/cm^2. This threshold is more than 2000 higher than the maximum TASER current density estimated in Mr. Lomax's heart.

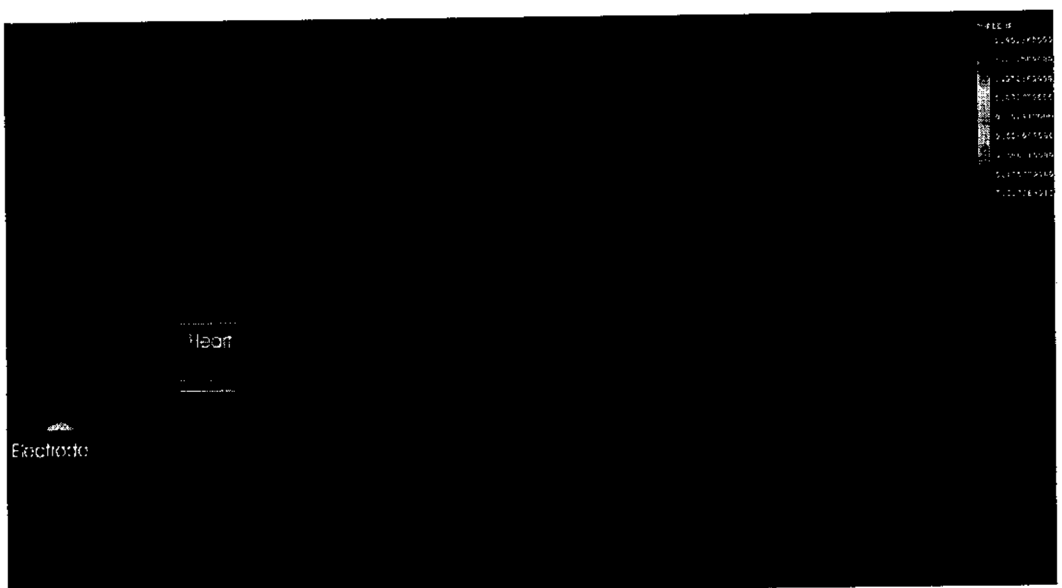

Figure 4. Current density distribution through the model [A/cm^2]. The current density decreases rapidly with distance from electrode. Only insignificant levels reach the heart.

These results are consistent with recent animal research reports that show TASER devices could not induce VF in swine [35]. Lakkireddy *et al.* studied five TASER electrode locations. Dr. Lakkireddy reported that no metabolic or hemodynamic changes were measured after X26 TASER discharge in any of the animals. Additionally, at standard TASER discharge levels, no VF episodes occurred. The FE modeling results presented above and the referenced research studies support data discussed in section 3.2.2 and indicated with almost certitude that insignificant, if any, TASER currents reached Mr. Lomax's heart during his confrontation with LVMPD officers. If any

TASER currents reached his heart, their magnitude would have been insufficient to trigger any kind of cardiac rhythm disturbances.

3.5 Effects of TASER ECDs electric fields on Nerve and Muscle

It is important to understand the electrical attenuating effects of the skin, fat and skeletal muscle layers. Additionally, speculation has been raised that TASER currents could produce skeletal muscle or nerve damage. The models analyzed herein describe skeletal muscle and motor nerve activation, cell electroporation and current and electric field distributions.

In general, skeletal muscle activation by electrical stimulation is elicited by excitation of α-motor neurons which innervate such muscle fibers. This fact often comes as a surprise, in that skeletal muscle cells are themselves excitable. Skeletal muscle excitability, however, is less than that of motor neuron cells in that both rheobase and chronaxie values of skeletal muscle are higher than those of the myelinated nerve axons which innervate them. To be effective in producing temporary incapacitation, each TASER pulse has to inject enough current to capture sufficient volume of skeletal muscle, through indirect stimulation via motor nerves. At the same time, to avoid direct tissue damage, the current densities (J) and electric field strengths (E) have to be lower than, for example, thresholds that may produce cell electroporation. Based on existing modeling and experimental literature, I have assumed the following J and E thresholds for excitation [36-39]:

- Motor neurons: chronaxie ~ 140 μs, rheobase E field ~ 0.06 to 0.15 V/cm for excitation at axon terminations such as motor end-plates;
- Strength-duration correction of needed E field strength for the ADVANCED TASER M26: (1 + 140/10)x(0.06 to 0.15 V/cm) = 0.9 to 2.25 V/cm
- Strength-duration correction of needed E field strength for the TASER X26: (1 + 140/70)x(0.06 to 0.15 V/cm) = 0.18 to 0.45 V/cm

Gehl et al. reported that for irreversible electroporation field strengths of 1600 V/cm are necessary, respectively [37].

Based on these values, it is estimated that the TASER E field required to successfully activate motor nerves has to exceed 0.18-2.25 V/cm, whereas to avoid electroporation E has to be less than 1600 V/cm. This yields a worst-case range for the E field strength of 2.25-1600 V/cm, to insure successful activation with either device while also avoiding electroporation. Figure 5 shows the electric field distribution in neck-area planes parallel to the electrode plane described in Figs 2-4 above. Per Officer Rader, in all situations he deployed the TASER electrodes in drive-stun mode in the general collar bone area [3]. Electric field values are listed in V/cm. The maximum E field values, 427 V/cm, are reached immediately underneath electrodes. The field then decreases rapidly with distance from electrodes. The two outer parallel planes are 2 cm on each side of the electrode plane. Even the maximum E field value is more than three times lower than values that produce irreversible tissue damage by electroporation. Note that E field values in the outer planes are significantly lower, less than 10-30 V/cm. While these values would be sufficient to capture motor nerves in immediate vicinity, they would be grossly insufficient to produce any nerve or muscle damage. It is also important to understand the

relationship of the phrenic nerve location (which originates from the C3 cervical area) in correspondence to TASER electrode locations on the surface of Mr. Lomax's neck.

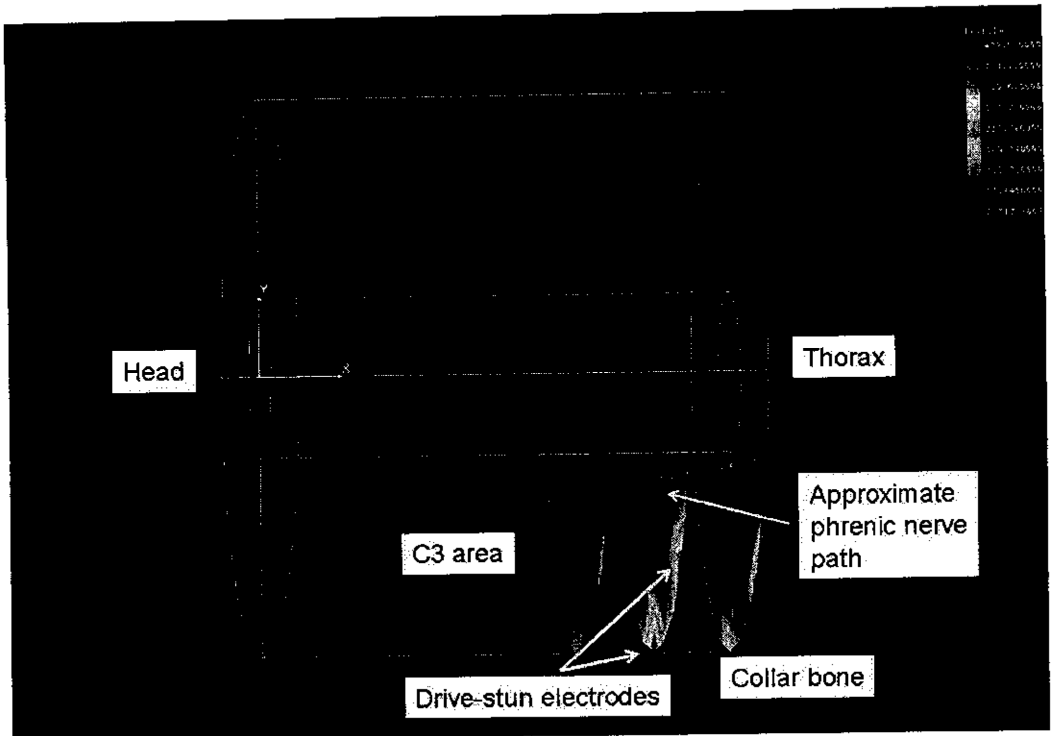

Figure 5. Estimation of electric field distribution in Mr. Lomax's neck area [V/cm]. E fields are insufficient to cause damage and have a very low probability of reaching to the phrenic nerve.

Figure 6 shows a typical cervical vertebra [40]. The phrenic spreads to the lower body through vertebral terminals similar to those shown in Fig. 6. Note that the nerve exits face away from the spinal protrusion next to the neck skin. Bone, unlike many other tissues, has a very low electrical conductivity. Additionally, as shown in Fig. 7, the phrenic nerve trajectory through the neck comes protected by other tissues, such as a significant layer of skin, fat and muscle, and goes at an angle away from the skin and towards the center axis of the body [40]. Corroborating this typical phrenic nerve path with the E field distribution in Fig. 5, the vertebral bone and other tissues would have blocked significant, perhaps all, current density from reaching to Mr. Lomax's phrenic nerve. Figure 5 shows that only very low electric field strength values reach out at the distance of the phrenic nerve. As described in [36] and in Table I, the skin, fat and skeletal muscle significantly attenuate TASER currents. The results in Table I were obtained by FE analyses that accounted for drive-stun electrode locations. However, the fat layer thickness was considered to be 3-5 mm. Given Mr. Lomax's obese configuration, it is very likely that the fat layer in his neck area was thicker than 3-5 mm. As such, it is highly likely that an additional level of electric field attenuation was provided.

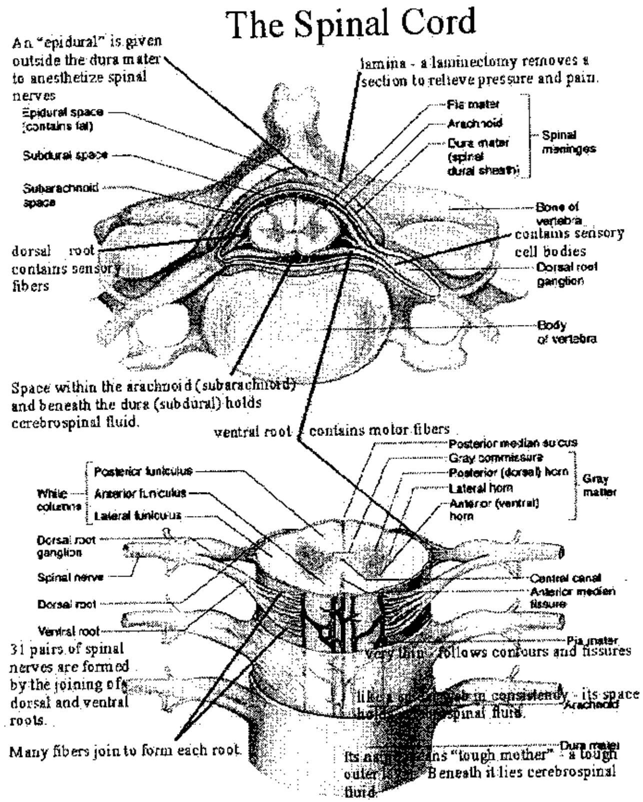

Figure 6. Typical structure of a cervical vertebra. Its bone structure offers significant electric shielding so that TASER currents are blocked from reaching nerve exits.

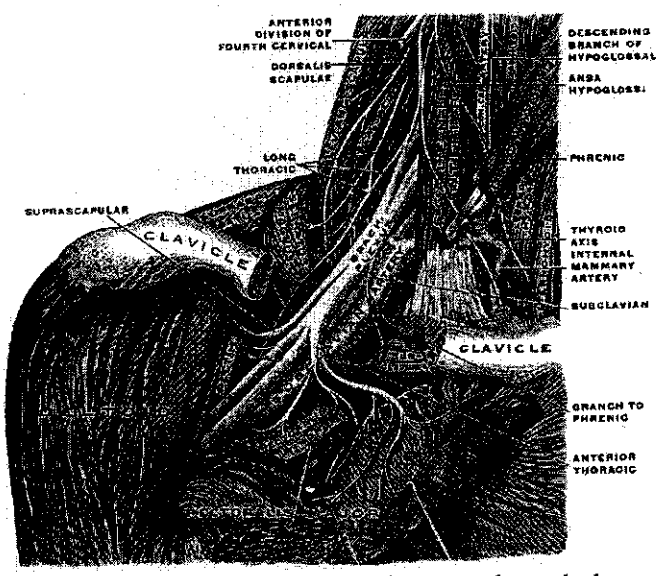

Figure 7. Typical trajectory of phrenic nerve through the neck.

Table I. Electrical Shell Effect of Fat and Skeletal Muscle

Condition	Jtrans [mA/cm2]	Jlong/Jtrans	Comments
Thin body with 5-mm fat and anisotropic muscle layers	15.63	8	Current is diverted away from deeper tissue layers by fat and longitudinal muscle electrical conduction
Muscle anisotropy removed	20.81	5	Removing muscle anisotropy increases current into deeper tissue layers by 30%
Fat and muscle anisotropy removed	45.49	2.9	Removing fat increases current into deeper tissue layers by 200%

Figure 8 shows the distribution of E field through a stack of tissues: skin, dermis, fat, muscle and body. Consistent with data in Table I, Fig. 8 illustrates that most of E field strength is shielded by the fat layer and by the anisotropy effects of skeletal muscle. Very little E field strength reaches below the skeletal muscle layer, into deeper layers of tissue.

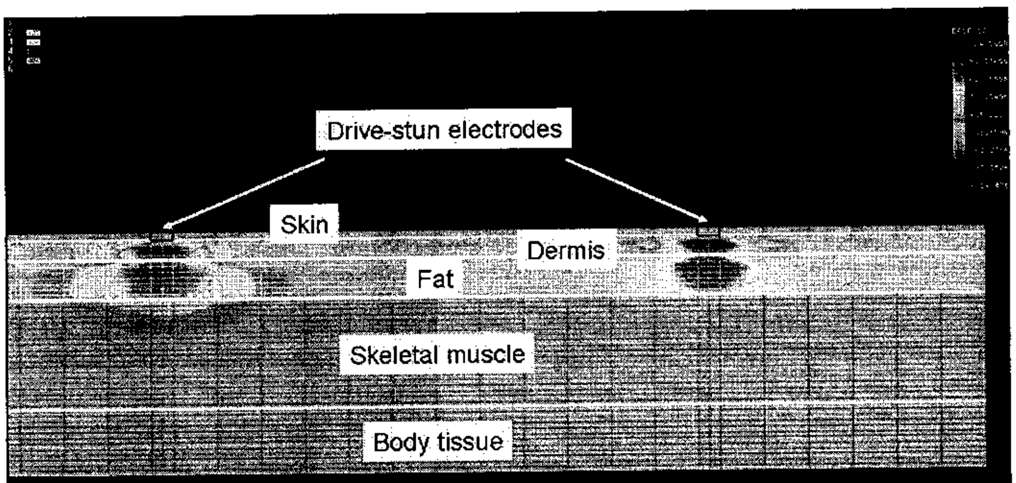

Figure 8. The high resistivity of fat and the anisotropy of skeletal muscle shield deeper layers of body tissue from TASER electric fields.

In conclusion, with a high degree of medical probability, TASER ECDs deliver currents that efficiently capture neuromuscular structures. In Mr. Lomax's case, it is highly probable that worst-case maximum values for TASER J and E are lower, by a very large factor, than levels reported to produce permanent cellular electroporation or nerve

and muscle damage. The phrenic nerve is protected by neighboring vertebral bone and overlying skin, fat and skeletal muscle against any significant TASER electric fields.

3.6 Delayed ventricular fibrillation allegations

The concept of delayed triggering of ventricular fibrillation (VF) has been circulated in the past. Blackwell and Hayllar conducted a 3-year prospective audit of 212 presentations to the emergency department after electrical injury [41]. After four years of follow-up, per their protocol, Blackwell and Hayllar showed that from their 212-patient pool no reported death or representation of abnormal cardiac rhythm have occurred as a result of the patients' electrical injury [41]. Based on Blackwell and Hayllar results, I conclude that the concept of delayed VF is not substantiated by scientific evidence and that it is virtually guaranteed that Mr. Lomax did not experience a delayed VF episode that might have been caused by the X26 TASER use.

3.7 Probable effects of phencyclidine overdose

Phencyclidine (PCP) is a dissociative anesthetic that was originally synthesized for intravenous use in 1957. If abused, PCP may cause serious adverse reactions, such as extreme agitation, high blood pressure (including complications, such as intracerebral complications), mild tachycardias, cardiac muscle fibrosis, psychosis, nystagmus, tachypnea and irregular respiratory, bronchorrhea, rhabdomyolysis, muscle rigidity, dystonia, opisthotonos, torticollis, akinesis, and hyperkinesis [42-44]. Mr. Lomax had a documented history of PCP and cocaine abuse. On the day of his death, he reportedly exhibited most of the symptoms and conditions listed above [1-3]. For example, on February 20, 2004, at 20:51, his rhythm was mildly tachycardic, documented at 123 bpm, his systolic blood pressure was mildly elevated at 141 mmHg [6]. His medical history records indicated prior abuse of PCP with similar symptoms that required medical intervention [7].The autopsy report concluded that Mr. Lomax "died as a result of cardiac arrest during restraining procedures. Phencyclidine intoxication and bronchopneumonia were significant contributing conditions."[8] Mr. Lomax's PCP concentration levels were measured as high as 245 ng/ml [8]. A PCP dose of 1000 ng/ml is considered lethal [42-44]. Additionally, the report found "foci of increased interstitial cardiac fibrosis", consistent with reported effects of PCP [8, 42-44]. Consistent with the autopsy and toxicology reports and with reported symptoms and conditions, Mr. Lomax's high concentrations of PCP seem highly likely to have been contributors to his demise.

3.8 Comments on Dr. Bush's expert report for plaintiff

Dr. Bush's report stated that 8% of PCP intoxications result in fatalities [45]. The autopsy report concluded that PCP was a significant contributor to Mr. Lomax's death [8]. It spite of his statement and the autopsy evidence, Dr. Bush rushed to dismiss PCP as having a proximal role in Mr. Lomax's death. In downplaying the role of PCP, Dr. Bush provided no analyses to elucidate scientifically whether or not Mr. Lomax's reported symptoms, such as mild tachycardia, high blood pressure, cardiac fibrosis, extreme

agitation, loss of orientation, respiratory difficulties, were related to his abuse of PCP. As discussed above, all these symptoms and conditions are known effects of PCP abuse [42-44]. Dr. Bush's decision to ignore addressing them makes his conclusion questionable.

3.9 Comments on Dr. Strote's expert report for plaintiff

Dr. Strote's report stated that the TASER currents may have disrupted Mr. Lomax's respiratory system [45]. Dr. Strote went to speculate that stun mode application in the neck area caused contraction of accessory muscles. However, he did not present evidence for his statement. The finite element analyses presented in sections 3.2-3.5 above showed that the temporary incapacitation achieved by TASER devices did not involve direct muscular capture. Rather, TASER currents captured local motor nerves that in turn caused loss of neuromuscular control, with the initial reaction being a gravitational dysreflexia. Additionally, note that even the autopsy report does not list any evidence of no musculoskeletal abnormalities (e.g. as in those that could have been theoretically produced by too strong electric fields), no damage to anterior neck muscles, no focal areas of pathological changes to the central nervous system (CNS) structures, unremarkable brain vessels, no CNS hemorrhage [8]. As such, Dr. Strote assumption that TASER discharge captured respiratory accessory muscles is speculative and not backed by scientific evidence.

3.10 Comments on Dr. Woodard's expert report for plaintiff

Dr. Woodard's report states that TASER application worsened Mr. Lomax's already present metabolic derangement [47]. However, Dr. Woodard does not provide any scientific quantification for the amount of additional derangement. Note that the autopsy report particularly states that, notwithstanding pulmonary edema, Mr. Lomax's musculoskeletal system was intact, no focal areas of pathological changes to CNS structures [8]. Consequently, his conclusion that the TASER application contributed to Mr. Lomax's expiration is vague, made without scientific backing and speculative.

3.11 Comments on Dr. Rhode's expert report for plaintiff

Dr. Rhode's report shows a misunderstanding of the "skin effect" as applied to TASER currents [48]. As shown above and in reference [36], in the case of TASER currents, the "skin effect" consists of the electrical attenuation provided by superficial layers of tissues, such as dermis and fat, and by the electrical anisotropy of the skeletal muscle. These layers significantly attenuate the magnitude of TASER electric fields. As such, I consider Dr. Rhode's statements on "skin effects" highly inaccurate. Furthermore, Dr. Rhodes states that "It is my opinion that the Taser current pulses can and do damage nerve cells ..." This strong opinion is not supported by any scientific evidence that Dr. Rhode provides in his report. He does not cite any scientific publication nor does he provide any scientific computations or numerical analyses of his own. As I have shown in sections 3.2 – 3.5 above, with a high degree of medical probability, TASER electric fields that reach into the skeletal neuromuscular layers are by a high margin lower than

thresholds reportedly known to produce permanent damage. The analyses present above are worst-case and it is highly likely that in Mr. Lomax's case the electrica field attenuation was even higher due to his obese conformation. Therefore, Dr. Rhode again makes speculative and gratuitous statements that are not supported by scientific evidence. Dr. Rhode makes additional speculative statements regarding nerve damage produced by TASER currents. Particularly, Dr. Rhode speculates that TASER currents could produce phrenic nerve damage. Yet again, he provides no scientific base for his statements. Dr. Rhode uses his speculative nerve damage statements to make a point that TASER current caused Mr. Lomax to have a respiratory arrest which resulted in his death. Note that the autopsy report particularly states that, notwithstanding pulmonary edema, Mr. Lomax's respiratory system was intact, with no signs of arrest [8]. Additionally, no musculoskeletal abnormalities (e.g. as in those that could have been theoretically produced by too strong electric fields), no damage to anterior neck muscles and no focal areas of pathological changes to the CNS structures were found [8]. The electric field analyses presented above (in association with Figs. 5-8 and Table I) show that, with a high degree of medical probability, phrenic nerve damage is very unlikely with use of TASER devices. By corroborating the electric field analyses with the autopsy results, which show no evidence of phrenic nerve damage, I infer that Dr. Rhode's statements are incorrect and his conclusion is baseless, unfunded speculation. Another gratuitous statement of Dr. Rhode relates to the delayed effects electric shocks may have on organs. He attempts to persuade the reader of his report that such effects are real and supported by prior publications. However, Dr. Rhode provides no references that show support of his delayed damage theory. In fact, as presented in section 3.6 above, long-term follow-up studies of patients that suffered confirmed electrical injuries showed no evidence of any delayed critical or fatal consequences [41]. Consequently, Dr. Rhode's statement regarding delayed organ failures is highly likely incorrect, certainly not supported by any medical evidence.

3.12 Opinions

3.12.1 The X26 TASER device is efficacious in producing suspect's temporary incapacitation, yet, per their intended use, they are safe and unlikely, with a high degree of medical probability, to directly cause fatal cardiac arrhythmias, permanent or serious muscular or nerve damage.

3.12.2 The concept of delayed ventricular fibrillation following electrical injury is not backed by any scientific evidence.

3.12.3 With a high degree of medical probability, the X26 TASER currents did not cause or contributed to Mr. Lomax's death. Rather, Mr. Lomax documented PCP abuse and overdose coupled with the physical effort caused by his fight with and aggression on housing and LVMPD officers are highly likely to be main contributors to his demise.

3.12.4 In spite of their solid resumes, the plaintiff's expert witnesses did not use scientific evidence in their reports. Drs. Bush, Strote, Woodard and Rhode are using speculative argumentation, rather than science, to arrive to their respective conclusions. As such, in my opinion, their final statements and conclusions are incorrect.

Prepared by Dorin Panescu, Ph.D. April 12, 2007

Appendix A: Dorin Panescu's CV

EXPERIENCE

Principal Staff Scientist, Cardiac Rhythm Management, St. Jude Medical, Sunnyvale, CA 2005 – present
Development of implantable medical devices for cardiac rhythm and heart failure control

Vice President, Research and Development, Refractec, Irvine, CA 2004 – 2005
Design, development and manufacturing of electrical medical devices for ophthalmic surgery

Senior Director, Systems Development, Boston Scientific, San Jose, CA 1996 – 2004
Design and development of electrical medical devices for cardiac radiofrequency ablation

Electrical safety testing of cardiac ablation and mapping devices

Senior R&D Engineer, EP Technologies, Sunnyvale, CA 1993 – 1995
Design and development of electrical medical devices for cardiac radiofrequency ablation

Electrical safety testing of cardiac ablation and mapping devices

Consultant, University of Wisconsin-Madison, Dept. of Electrical Engineering. 1996 – present
Cardiac and liver radiofrequency ablation research

Research Assistant, University of Wisconsin-Madison. 1991 – 1993
Finite element electrical modeling of transthoracic and implantable defibrillators and pacemakers

Development of a database of ventricular fibrillation episodes, Dane County, Wisconsin

Teaching Assistant, University of Wisconsin-Madison. Spring 1991
Nonlinear electronic circuits

R & D Engineer, Institute for Automation, Romania. 1989 - 1990
Development of microprocessor-controlled data acquisition systems

Teaching Assistant, University of Cluj, Romania. 1989 - 1990
Analog and digital circuits

Production Manager, IAEM (division of ABB), Romania. 1985 - 1989
Manufacturing of electronic temperature regulators

EDUCATION

Ph.D. Electrical and Computer Engineering, University of Wisconsin-Madison,	August 1993.
M.S. Electrical and Computer Engineering, University of Wisconsin-Madison	December 1991
B.S. Electronics and Telecommunications, Polytechnic Institute of Timisoara, Romania.	June 1985

INVITED LECTURES

Seven invited domestic and international lectures related to pacing, defibrillation and cardiac ablation

PATENTS

Inventor or co-inventor on over 125 issued US patents related to cardiac mapping, ablation and imaging

PUBLICATIONS

Author or co-author on over 90 technical publications related to cardiac pacing, cardiac defibrillation, cardiac radiofrequency ablation, cardiac imaging, analog and digital circuit design, digital signal processing

AWARDS

2003 - Patent Milestone Award – Boston Scientific
2002 – IEEE-EMBS Early Career Achievement Award
2002 - Patent Milestone Award – Boston Scientific
2001 - John Abele Science and Technology Award – Boston Scientific
2001 - Patent Milestone Award – Boston Scientific
1982 – "Traian Lalescu" Award – Polytechnic Institute of Timisoara, Romania

PROFESSIONAL AFFILIATION

Fellow, American Institute of Medical and Biological Engineering
Senior Member, Institute of Electrical and Electronics Engineers (IEEE)
Member, IEEE Engineering in Medicine and Biology Society (EMBS)

Served as:

2005-2008 Chair, Therapeutic Systems and Technologies Technical Committee, IEEE-EMBS
2005 Chair of Industry Relations Committee IEEE-EMBS
2004 Co-chair of the Cardiovascular Systems Tracks at the IEEE–EMBS Conference, San Francisco
2003 Co-chair of the Cardiovascular Systems Tracks at the IEEE–EMBS Conference, Cancun, Mexico
2002 IEEE-USA Medical Technology Committee
1997-1998 Member of the IEEE-EMBS AdCom
1997-1998 IEEE-EMBS Region 6 Representative
1997 Co-chair of the Cardiovascular Systems Theme at the IEEE–EMBS Conference, Chicago.
1997-1998 Chair of Industry Relations Committee IEEE-EMBS

SELECTED LIST OF ISSUED PATENTS (out of over 170 issued US patents and published US patent applications)

7194294/2007	Multi-functional medical catheter and methods of use. Inventors: D. Panescu, and D. K. Swanson
6895267/2005	Systems and methods for guiding and locating functional elements on medical devices positioned in a body. Inventors: D. Panescu, D. W. Arnett and D. K. Swanson
6790206/2004	Compensation for power variation along patient cables. Inventors: D. Panescu
6746401/2004	Tissue ablation visualization. Inventors: D. Panescu
6735465/2004	Systems and processes for refining a registered map of a body cavity. Inventors: D. Panescu
6428536/2002	Expandable-collapsible electrode structures made of electrically conductive material. Inventors: D. Panescu, D. K. Swanson, J. G. Whayne and T. F. Kordis
6370435/2002	Systems and methods for examining the electrical characteristic of cardiac tissue. Inventors: D. Panescu, D. K. Swanson, M. S. Mirotznik, D. S. Schwartzman and K. R. Foster
6293943/2001	Tissue heating and ablation systems and methods which predict maximum tissue temperature. Inventors: D. Panescu, S. D. Fleischman and D. K. Swanson
6289239/2001	Interactive systems and methods for controlling the use of diagnostic and therapeutic instruments in interior body regions. Inventors: D. Panescu, D. McGee, J. G. Whayne, R. R. Burnside, D. K. Swanson and D. A. Dupree
5925038/1999	Expandable-collapsible electrode structures for capacitive coupling to tissue. Inventors: D. Panescu, D. K. Swanson, J. G. Whayne and T. F. Kordis
5810802/1998	Systems and methods for controlling tissue ablation using multiple temperature sensing elements. Inventors: D. Panescu, S. D. Fleischman, J. G. Whayne and D. K. Swanson
5755715/1998	Tissue heating and ablation systems and methods using time-variable set point temperature curves for monitoring and control. Inventors: R. A. Stern, D. Panescu and D. K. Swanson
5688267/1997	Systems and methods for sensing multiple temperature conditions during tissue ablation. Inventors: D. Panescu, D. K. Swanson, S. D. Fleischman and T. M. Bourne
5487391/1996	Systems and methods for deriving and displaying the propagation velocities of electrical events in the heart. Inventor: D. Panescu
5485849/1996	System and methods for matching electrical characteristics and propagation velocities in cardiac tissue. Inventors: D. Panescu and D. K. Swanson

SELECTED LIST OF PUBLICATIONS (out of over 100)

Books

1. D. Panescu, "Medical Device Industry," in M. Akay, Ed., *Wiley Encyclopedia of Biomedical Engineering*, Hoboken, NJ: Wiley & Sons, 2006.
2. D. Panescu, "Other Time– and Frequency– Domain Techniques," in W. J. Tompkins, Ed., *Biomedical Digital Signal Processing: C-Language Examples and Laboratory Experiments for the IBM PC*, Englewood Cliffs, NJ: Prentice-Hall, 1993.

Journal Articles

3. D. Panescu,, "Less-than-lethal weapons: Design and Medical Safety of Neuromuscular Incapacitation Devices," *IEEE Eng Med Biol Mag.*, vol. 26(4), 2007.
4. D. Panescu,, "MEMs in Medicine and Biology," *IEEE Eng Med Biol Mag.*, vol. 25(5), pp. 19-28, 2006.
5. D. Panescu,, "Healthcare Applications of RF Identification," *IEEE Eng Med Biol Mag.*, vol. 25(3), pp. 77-83, 2006.
6. D. Panescu,, "Vagus Nerve Stimulation for the Treatment of Depression," *IEEE Eng Med Biol Mag.*, vol. 24(6), pp. 68-72, 2005.
7. D. Panescu,, "An imaging pill for gastrointestinal endoscopy," *IEEE Eng Med Biol Mag.*, vol. 24(4), pp. 12-14, 2005.
8. D. Panescu,, "Cardiac Resynchronization Therapy," *IEEE Eng Med Biol Mag.*, vol. 24(2), pp. 22-26, 2005.
9. D. Panescu,, "Conductive Keratoplasty," *IEEE Eng Med Biol Mag.*, vol. 23(4), pp. 16-18, 2004.
10. D.S. Khoury, L. Rao, C. Ding, H. Sun, K.A. Youker, D. Panescu and S.F. Nagueh, "Localizing and quantifying ablation lesions in the left ventricle by myocardial contrast echocardiography," *J Cardiovasc Electrophysiol.*, vol. 15(9), pp. 1088-1090, 2004.
11. D. Panescu,, "Seeing clearly with Refractec, Inc.," *IEEE Eng Med Biol Mag.*, vol. 23(3), pp. 8-12, 2004.
12. D. Panescu,, "Drug Eluting Stents," *IEEE Eng Med Biol Mag.*, vol. 23(2), pp. 21-23, 2004.
13. D. Panescu, S. D. Fleischman, J. G. Whayne, D. K. Swanson, M. S. Mirotznik, I. McRury and D. E. Haines, "Radiofrequency multielectrode-catheter ablation in the atrium," *Phys. Med. Biol.*, vol. 44, pp. 899-915, 1999.
14. I. D. McRury, D. Panescu, M. A. Mitchell and D. E. Haines, "Non-uniform heating during catheter ablation with long electrodes: Monitoring the edge effect," *Circulation*, vol. 96, pp. 4057–4064, 1997.
15. D. Panescu, "Intracardiac mapping and radiofrequency catheter ablation for the therapy of ventricular tachycardia," *Physiol. Meas.*, vol. 18, pp. 1–38, 1997.
16. D. Panescu, J. G. Webster, W. J. Tompkins and R. A. Stratbucker, "Optimization of transcutaneous cardiac pacing by three-dimensional finite element modeling of the human thorax," *Med. Biol. Eng. Comput.*, vol. 33(6), pp. 769-775, 1995.
17. D. Panescu, J. G. Whayne, S. D. Fleischman, M. S. Mirotznik, D. K. Swanson and J. G. Webster, "Three-dimensional finite element analysis of current density and of thermal profiles during radiofrequency ablation," *IEEE Trans. Biomed. Eng.*, vol. 42, no. 9, pp. 879–890, 1995.
18. D. Panescu, J. G. Webster, W. J. Tompkins and R. A. Stratbucker, "Optimization of cardiac defibrillation by three-dimensional finite element modeling of the human thorax," *IEEE Trans. Biomed. Eng.*, vol. 42, no. 2, pp. 185–192, 1995.
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23. D. Panescu, J. G. Webster and R. A. Stratbucker, "Modeling current density distribution during transcutaneous cardiac pacing," *IEEE Trans. Biomed. Eng.*, vol. 41, no. 6, pp. 549–555, 1994.
24. D. Panescu, J. G. Webster and R. A. Stratbucker, "Measurement of ventricular volume from blood conductance using two-dimensional finite element analysis," *Physiol. Meas.*, vol. 15(1), pp. 49–56, 1994.
25. D. Panescu, K. P. Cohen, J. G. Webster and R. A. Stratbucker, "The mosaic electrical characteristics of the skin," *IEEE Trans. Biomed. Eng.*, vol. 40, no. 5, pp. 434-439, 1993.

Appendix B: List of Past Expert Testimony in previous four years and Expert Fee Rate Sheet.

In the last 4 years, I have not testified as an expert at trial or by deposition. I have provided expert witness reports for the following cases:

1. The Estate of James Borden vs. TASER International, Inc.
2. The Estate of Keith Tucker vs. TASER International, Inc.
3. Lewis vs. TASER International, Inc.
4. The Estate of Woolfolk vs. TASER International, Inc.
5. The Estate of Richard Holcomb vs. TASER International, Inc.
6. TASER International, Inc. vs. James A Ruggieri
7. Heston vs. TASER International, Inc.
8. King vs. TASER International, Inc.

I charge $135/hour for expert witness work and report preparation and $270/hour for deposition and court appearances.

Sincerely,

Dorin Panescu, Ph.D.